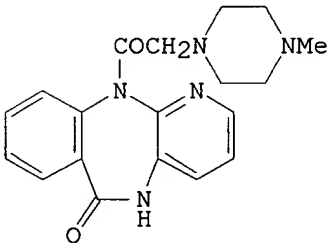
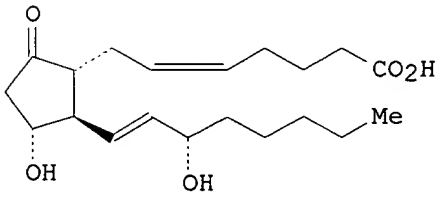
AN 1982:174175 CAPLUS
DN 96:174175
TI Study on the cytoprotective properties of pirenzepine, PGE2, and cimetidine in rats
AU Del Soldato, P.
CS Ist. De Angeli S.p.A., Milan, Italy
SO Boll. Chim. Farm. (1981), 120(11), 631-8
CODEN: BCFAAI; ISSN: 0006-6648
DT Journal
LA Italian
CC 1-9 (Pharmacology)
Section cross-reference(s): 2
GI



I



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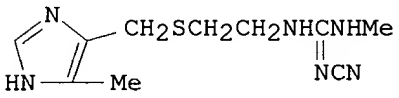
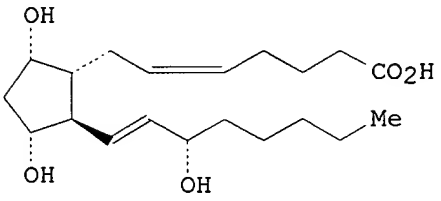


III

AB pirenzepine (I) [28797-61-7] and PGE2 (II) [363-24-6], but not cimetidine (III) [51481-61-9], protected the gastric mucosa of rats in vivo against damage from EtOH, taurocholic acid plus HCl, or acetylsalicylic acid plus HCl. The effect of both drugs was dose-dependent. I was effective when given either prophylactically or therapeutically, whereas II was active only in the former mode. II is considered to act on the mucosa at the cellular or tissue level, whereas the effect of I is mainly submucosal.
ST stomach protection pirenzepine PGE2 cimetidine
IT Stomach, toxic chemical and physical damage
(mucosa, damage to, cimetidine and PGE2 and pirenzepine protection against)
IT 363-24-6 28797-61-7 51481-61-9
RL: BIOL (Biological study)
(stomach mucosa damage inhibition by)

AN 1982:521114 CAPLUS
DN 97:121114
TI The effect of PGF2.alpha. on human oral mucous membrane
AU Terner, Kornelia; Javor, T.
CS Ist Dep. Med., Univ. Med. Sch., Pecs, Hung.
SO Pharmacol. Res. Commun. (1982), 14(6), 511-22
CODEN: PLRCAT; ISSN: 0031-6989
DT Journal
LA English
CC 2-9 (Mammalian Hormones)
GI



I

AB PGF2.alpha. (I) [551-11-1] topical treatment of oral tissues decreased or inhibited the caustic response to subsequent exposure to AgNO3 or trichloroacetic acid [76-03-9], indicating that I has some cytoprotective action on the human oral mucosa. Submucosal injection of I induced a mild vasodilation, but topically applied I had no effect on the mucous membrane. An inflammatory response occurred after injection of I and smearing of the tissue with caustics, but this disappeared in 15-30 min.

ST prostaglandin cytoprotection oral mucosa; PGF2 cytoprotection oral mucosa

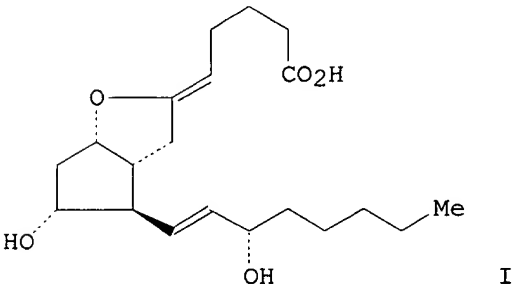
IT Mouth
(mucosa, PGF2.alpha. cytoprotective effect on)

IT 551-11-1
RL: BIOL (Biological study)
(mouth mucosa cytoprotection by)

IT 76-03-9, biological studies 7761-88-8, biological studies
RL: BIOL (Biological study)
(mouth mucosa toxic response to, PGF2.alpha. inhibition of)

AN 1982:557157 CAPLUS
DN 97:157157
TI Cytoprotection by PGE2, atropine, pirenzepine and vagotomy in rats
AU Rovati, V.; Foschi, D.; Ferrante, F.; Del Soldato, P.; Daniotti, S.; Varin, L.
CS "L. Sacco" Hosp., Univ. Milan, Milan, Italy
SO Scand. J. Gastroenterol., Suppl. (1982), 17(72), 261-4
CODEN: SJGSB8; ISSN: 0085-5928
DT Journal
LA English
CC 2-9 (Mammalian Hormones)
Section cross-reference(s): 14
AB Gastric cytoprotective effects of vagotomy, PGE2 (I) [363-24-6], and the antimuscarinic compds. pirenzepine [28797-61-7] and atropine [51-55-8] were studied in rats. Both pharmacol. and surgical treatment prevented the gastric damage induced by intragastric administration of acetylsalicylic acid + HCl. The mechanisms of action are discussed.
ST stomach cytoprotection PGE2 vagotomy antimuscarinic; ulcer antimuscarinic vagotomy PGE2
IT Ulcer
(antimuscarinics and PGE2 and vagotomy prevention of)
IT Stomach, toxic chemical and physical damage
(mucosa, aspirin damage of, antimuscarinic compds. and PGE2 and vagotomy protection against)
IT Nerve
(vagus, section of, gastric cytoprotection by)
IT 51-55-8, biological studies 363-24-6 28797-61-7
RL: BIOL (Biological study)
(stomach cytoprotection by)

AN 1982:593728 CAPLUS
DN 97:193728
TI PGI2 prevents ischemia-induced alterations in cardiac catecholamines without influencing nerve stimulation-induced catecholamine release in nonischemic conditions
AU Schroer, Karsten; Darius, Harald; Addicks, Klaus; Koester, Rolf; Smith, Edward F., III
CS Pharmakol. Inst., Univ. Koeln, Cologne, Fed. Rep. Ger.
SO J. Cardiovasc. Pharmacol. (1982), 4(5), 741-8
CODEN: JCPCDT; ISSN: 0160-2446
DT Journal
LA English
CC 2-9 (Mammalian Hormones)
Section cross-reference(s): 14
GI



I

AB Acute myocardial ischemia was produced in rabbit Langendorff hearts by ligation of the left anterior descending coronary artery for 2 h. This was accompanied by an increase in creatine kinase [9001-15-4] activity of the ischemic myocardium as compared to sham-operated nonischemic controls and by a decrease in ATP [56-65-5] levels from 2.25 mol/g wet wt. in the nonischemic area to 0.95 mol/g wet wt. in the ischemic area, indicating a considerable degree of tissue damage. There was a decrease in the norepinephrine [51-41-2] ratio between infarcted and noninfarcted myocardium from 1.08 in sham-operated controls to 0.66 in ischemic hearts. Histochem. revealed a nearly complete loss of fluorescence in perivascular adrenergic nerves in the ischemic area. Infusion of prostacyclin (PGI2)(I) [35121-78-9] (1.1 nmol/min), starting 10 min prior to ischemia, abolished the increase in creatine kinase activity and the decrease in ATP levels of the ischemic myocardium. I also prevented the ischemia-induced alterations in catecholamine content and the decrease in adrenergic fiber fluorescence. I did not influence myocardial dynamics and O2 consumption. To det. the effect of I on nerve stimulation-induced catecholamine release, a sep. group of Langendorff rabbit hearts with intact right sympathetic nerves was stimulated twice for 1 min at 0 and 13 min. I at 30 nM=3 .mu.M had no effect on catecholamine overflow when compared to control hearts. Evidently, I exerts a stabilizing effect on cell membranes that prevents ischemia-induced destruction of adrenergic nerve endings. This cytoprotective effect is restricted to the ischemic area and does not interfere with the physiol. nerve stimulation-induced norepinephrine release.

ST heart ischemia cytoprotection PGI2; catecholamine heart ischemia PGI2
IT Catecholamines
RL: BIOL (Biological study)
(of heart ischemic myocardium, PGI2 effect on)
IT Heart, disease or disorder

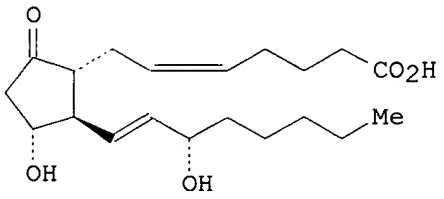
(ischemia, PGI2 cytoprotective action in, ATP and catecholamines and creatine kinase in relation to)

IT 35121-78-9
RL: BIOL (Biological study)
(heart ischemic myocardium protection by, ATP and catecholamines and creatine kinase contents in relation to)

IT 51-41-2 56-65-5, biological studies 9001-15-4
RL: BIOL (Biological study)
(of heart ischemic myocardium, PGI2 effect on)

=>

AN 1982:609343 CAPLUS
DN 97:209343
TI Influence of prostaglandin on actinomycin C-induced degeneration of embryonal neuroectodermal tissue
AU Stachura, Jerzy; Kaluza, Jozef
CS Inst. Pathol., N. Copernicus Med. Acad., Krakow, 31531, Pol.
SO Prostaglandins (1982), 24(3), 433-40
CODEN: PRGLBA; ISSN: 0090-6980
DT Journal
LA English
CC 2-9 (Mammalian Hormones)
GI



I

AB The protective action of PGE2 (I) [363-24-6] (0.02-0.05M) on actinomycin C (AMC) [8052-16-2]-induced degeneration of embryonal neuroectodermal tissue was examd. in vitro. Material for tissue culture was taken from the cerebrum and cerebellum of 12-day-old chick embryos. AMC was added to 7-day organotypic tissue cultures exhibiting a growth zone equal to the diam. of explant. Morphol. evaluation of the AMC-induced damage was performed after 24 h, including quantitation of degenerated and normal neuroectodermal cells. The AMC-induced degeneration of embryonal neuroectodermal tissue was reduced by I administration. This protective action of I was dose-dependent.

ST prostaglandin cytoprotection neural tissue; PGE2 embryo neuroectoderm cytoprotection

IT Brain, toxic chemical and physical damage
(actinomycin C degeneration of neuroectodermal tissue of, of embryo, PGE2 cytoprotective action on)

IT Nerve
(cytoprotection of, by prostaglandins)

IT Prostaglandins
RL: BIOL (Biological study)
(nerve tissue cytoprotection by)

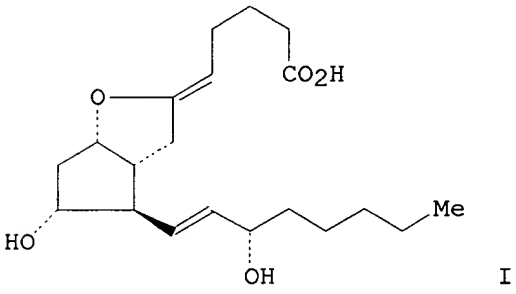
IT 363-24-6
RL: PROC (Process)
(cytoprotective action of, in nerve tissue)

IT 8052-16-2
RL: BIOL (Biological study)
(neuroectodermal degeneration induction by, in embryo, PGE2 protection against)

AN 1983:1006 CAPLUS
DN 98:1006
TI Effects of a dietary prostaglandin precursor on the progression of experimentally induced chronic renal failure
AU Barcelli, Uno O.; Weiss, Mark; Pollak, Victor E.
CS Med. Cent., Univ. Cincinnati, Cincinnati, OH, 45267, USA
SO J. Lab. Clin. Med. (1982), 100(5), 786-97
CODEN: JLCMAK; ISSN: 0022-2143
DT Journal
LA English
CC 2-9 (Mammalian Hormones)
Section cross-reference(s): 14
AB Normal linoleic acid (NLA) [60-33-3] or high linoleic acid (HLA) diets were pair-fed to groups of 3/4-nephrectomized and sham-operated rats. Serum creatinine and urinary protein excretion were measured serially. Nephrectomized rats on the NLA diet had progressive deterioration of renal function. By contrast, nephrectomized rats on the HLA diet maintained stable renal function. Urinary protein excretion was lower and glomerular sclerosis was prevented in the rats fed the HLA diet. No changes were obsd. in the levels of blood pressure, serum cholesterol, or serum triglycerides as an effect of the diet. Increased PGE2 [363-24-6] prodn., measured by RIA in the renal cortex of rats on the HLA diet, suggested that the protective effect on renal function in this model of chronic renal failure may be mediated by increased renal cortical PG formation.
ST linoleate diet kidney failure; prostaglandin kidney cortex linoleate cytoprotection
IT Prostaglandins
RL: FORM (Formation, nonpreparative)
(formation of, by kidney cortex, dietary linoleate stimulation of, in chronic renal failure)
IT Kidney, disease or disorder
(failure, chronic, linoleate effect on, in diet, prostaglandin formation in relation to)
IT 363-24-6
RL: FORM (Formation, nonpreparative)
(formation of, by kidney cortex, dietary linoleate stimulation of, in chronic renal failure)
IT 60-33-3, biological studies
RL: BIOL (Biological study)
(kidney failure response to dietary, prostaglandin formation in relation to)

AN 1983:12130 CAPLUS
DN 98:12130
TI Role of locally generated prostaglandins in adaptive gastric cytoprotection
AU Konturek, Stanislaw J.; Brzozowski, Tomasz; Piastucki, Ireneusz; Radecki, Tadeusz; Dembinski, Artur; Dembinska-Kiec, Aldona
CS Inst. Physiol. Pharmacol., Med. Acad., Krakow, Pol.
SO Dig. Dis. Sci. (1982), 27(11), 967-71
CODEN: DDSCDJ; ISSN: 0163-2116
DT Journal
LA English
CC 2-9 (Mammalian Hormones)
AB The role of mucosal generation of prostaglandins (PGs) in the ability of mild irritants (20% EtOH or 5% NaCl) to protect against the formation of mucosal lesions caused by necrotizing agents (100% EtOH or 25% NaCl) or acidified aspirin (ASA) was investigated. Mild irritants protected against damage from necrotizing agents but not from ASA. This protection was accompanied by increased mucosal generation of PGE2 [363-24-6] and PGI2 [35121-78-9] like substances. Exogenous PGE2 and PGI2 applied topically to the gastric mucosa in a nonantisecretory dose greatly inhibited the formation of lesions induced by either necrotizing agents or ASA. Pretreatment with indomethacin, which suppressed the generation of mucosal PGs augmented formation of lesions by necrotizing agents and partly counteracted the protective effect of mild irritants. Evidently, mild irritants, and exogenous PGs inhibit the formation of gastric lesions by necrotizing agents, at least in part, by mucosal generation of PGs.
ST stomach endogenous prostaglandin cytoprotection
IT Prostaglandins
RL: FORM (Formation, nonpreparative)
(formation of, by gastric mucosa, cytoprotective action of)
IT Ulcer
(inhibition of, prostaglandin formation in relation to)
IT Stomach, metabolism
(mucosa, prostaglandins formation by, cytoprotective action of)
IT 363-24-6 35121-78-9
RL: PROC (Process)
(stomach cytoprotective action of)
IT 50-78-2D, acidified
RL: BIOL (Biological study)
(stomach lesions from ,endogenous prostaglandins in relation to)

AN 1983:47648 CAPLUS
DN 98:47648
TI Cytoprotection of canine gastric mucosa by prostacyclin: possible mediation by increased mucosal blood flow
AU Konturek, Stanislaw J.; Robert, Andre
CS Inst. Physiol., Med. Acad., Krakow, 31-531, Pol.
SO Digestion (1982), 25(3), 155-63
CODEN: DIGEBW; ISSN: 0012-2823
DT Journal
LA English
CC 2-9 (Mammalian Hormones)
GI



I

AB The role of gastric mucosal blood flow (MBF) in the gastric cytoprotection produced by PGI2 (I) [35121-78-9] and PGE2 [363-24-6] was examd. in dogs. An acidified soln. of saline was applied topically on the canine gastric mucosa with and without EtOH at various concns. EtOH applied to the mucosa of a stomach flap prepn., in concns. of 5-80%, gradually decreased the transmucosal p.d. from -58 to -5 mV and increased net ionic fluxes. MBF gradually increased at lower concns. of EtOH, reaching the peak of .apprx.50% above basal at 20% EtOH and then declining at 40 and 80% EtOH. I (10 .mu.g/kg/h) prevented the changes in p.d. and ion fluxes produced by lower but not higher concns. of EtOH and this was accompanied by a marked increase in the MBF above the level produced by EtOH from -57 to -40 mV and elicited large net H+ and Na+ fluxes. MBF was increased by 30%. I (10 .mu.g/kg/h) completely prevented EtOH-induced changes in p.d., reduced ionic fluxes, and raised the MBF 2-fold. PGE2 (80 .mu.g/kg/h) did not affect EtOH-induced alterations in p.d., ion fluxes, and MBF. Thus, I, but not PGE2, effectively protects the canine gastric mucosa against the damage produced by EtOH. This cytoprotection may be due to increased mucosal circulation, which by an unknown mechanism interferes with the mucosal damage caused by EtOH.

ST stomach circulation prostacyclin cytoprotection

IT Stomach
(circulation of, PGI2 effect on, cytoprotective action in relation to)

IT Electric potential, biological
(of stomach mucosa, PGI2 effect on, cytoprotection in relation to)

IT Circulation
(of stomach, PGI2 effect on, cytoprotection in relation to)

IT Ulcer
(prostacyclin effect on stomach mucosa circulation and elec. potential in relation to)

IT 35121-78-9
RL: BIOL (Biological study)
(stomach circulation and elec. potential response to, in cyctoprotection)

IT 363-24-6
RL: BIOL (Biological study)
(stomach circulation in presence of, in ulcerogenic stimulus)

=>

AN 1995:613802 CAPLUS
DN 123:26219
TI Induction by prostaglandin A1 of heme oxygenase in myoblastic cells: an effect independent of expression of the 70 kDa heat shock protein
AU Rossi, Antonio; Santoro, M. Gabriella
CS Inst. Experimental Medicine, CNR, Rome, 00135, Italy
SO Biochem. J. (1995), 308(2), 455-63
CODEN: BIJOAK; ISSN: 0264-6021
DT Journal
LA English
CC 2-9 (Mammalian Hormones)
AB Prostaglandins of the A type (**PGA**) induce the synthesis of 70 kDa heat shock proteins (hsp70) in a large variety of mammalian cells. Induction of hsp70 has been assocd. with a **cytoprotective** effect of PGA1 after virus infection of thermal injury. In the present report the authors provide evidence that, in murine myoblasts, PGA1 is not able to induce hsp70 expression, whereas it increases the synthesis of the constitutive protein, hsc70, and dramatically induces the synthesis of a 32 kDa protein (p32). The p32 protein has been identified as heme oxygenase. PGA1 acts at the transcriptional level by inducing heme oxygenase mRNA synthesis, and the signal for induction appears to be assocd. with decreased intracellular GSH levels. Heme oxygenase, a low-mol. mass stress protein induced in mammalian cells by oxidant stress, is known to be part of a general inducible antioxidant defense pathway. The fact that prostaglandin synthesis is stimulated in muscle during contraction and in the heart in response to ischemia raises the possibility that induction of heme oxygenase by **PGA** in myoblasts could be part of a protective mechanism in operation during stress and hypoxia.
ST PGA1 heme oxygenase myoblast heatshock protein
IT Myoblast
(PGA1 stimulation of heme oxygenase in myoblast independent of hsp70)
IT Proteins, specific or class
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(hsp 70, PGA1 stimulation of heme oxygenase in myoblast independent of hsp70)
IT 70-18-8, GSH, biological studies
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(PGA1 stimulation of heme oxygenase in myoblast in relation to GSH attenuation)
IT **14152-28-4**, PGA1
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(PGA1 stimulation of heme oxygenase in myoblast independent of hsp70)
IT 9059-22-7, Heme oxygenase
RL: MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(PGA1 stimulation of heme oxygenase in myoblast independent of hsp70)

AN 1984:591508 CAPLUS
DN 101:191508
TI 5-Membered cyclic compounds, and their pharmaceutical use
IN Noyori, Ryoji; Fukushima, Masanori; Kurozumi, Seizi; Sugiura, Satoshi
PA Teijin Ltd. , Japan
SO Eur. Pat. Appl., 67 pp.
CODEN: EPXXDW
DT Patent
LA English
IC C07C069-738; C07C059-82; C07C177-00; C07C067-327; C07C051-00; A61K031-557; A61K031-19; A61K031-215
CC 26-3 (Biomolecules and Their Synthetic Analogs)
Section cross-reference(s): 1, 63
FAN.CNT 1

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	EP 106576	B1	19880309		
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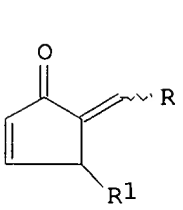
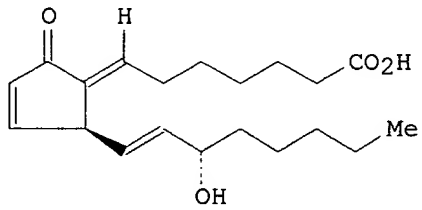


I



II

AB 7,8-Didehydro prostaglandin analogs (I, R, R1 = (un)substituted C1-12 aliph. hydrocarbon group] were prepd., by appropriate modification of conventional methods, and shown, in some cases, to have activity against Ehrlich ascites sarcoma and to be effective stomach **cytoprotective** agents at safer dosage levels than mytomycin C. Typical of compds. prepd. and tested was II.

ST prostaglandin antitumor stomach cytoprotectant
IT Neoplasm inhibitors
Neoplasm inhibitors
(7,8-didehydro prostaglandin analogs)
IT Stomach, disease or disorder
(**cytoprotective** agents for, 7,8-didehydro prostaglandin

analogs as)
IT Prostaglandins
RL: RCT (Reactant)
(analogs, 7,8-didehydro)
IT 56745-67-6
RL: RCT (Reactant)
(alkylation of, in synthesis of prostaglandin analogs)
IT 106-95-6, reactions
RL: RCT (Reactant)
(alkylation with, of **cyclopentenone** deriv.)
IT 123-72-8 35376-00-2
RL: RCT (Reactant)
(condensation of, with **cyclopentenone** deriv.)
IT 86982-76-5 86982-78-7 86982-88-9 92711-61-0 92711-63-2
92711-64-3 92762-27-1 92762-28-2 92762-29-3
RL: RCT (Reactant)
(dehydration of)
IT 86982-91-4P 92711-56-3P 92711-57-4P 92711-58-5P 92711-59-6P
92711-60-9P
RL: BAC (Biological activity or effector, except adverse); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation)
(prepn. and antitumor activity of)
IT 92711-65-4P 92711-66-5P 92711-67-6P 92711-68-7P 92711-69-8P
92711-70-1P 92711-71-2P 92711-72-3P 92711-73-4P 92711-74-5P
92711-75-6P 92711-99-4P 92712-00-0P 92731-71-0P 92762-30-6P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and dehydration of)
IT 86982-71-0P 86982-78-7P 86982-86-7P 92711-76-7P 92711-77-8P
92711-78-9P 92711-79-0P 92711-80-3P 92711-81-4P 92711-82-5P
92711-83-6P 92711-84-7P 92711-95-0P 92762-31-7P 92762-32-8P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and deprotection of)
IT 86982-86-7P 92711-85-8P 92711-86-9P 92711-87-0P 92711-88-1P
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92711-94-9P 92711-96-1P 92711-97-2P 92711-98-3P
RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. of)
IT **92711-55-2P** 92762-26-0P
RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn., and antitumor and **cytoprotective** activities of)
IT 92711-62-1P
RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn., dehydration, amd deprotection of)
IT 60220-91-9
RL: RCT (Reactant)
(use of, in synthesis of prostaglandin analogs)

AN 1995:613802 CAPLUS
DN 123:26219
TI Induction by prostaglandin A1 of heme oxygenase in myoblastic cells: an effect independent of expression of the 70 kDa heat shock protein
AU Rossi, Antonio; Santoro, M. Gabriella
CS Inst. Experimental Medicine, CNR, Rome, 00135, Italy
SO Biochem. J. (1995), 308(2), 455-63
CODEN: BIJOAK; ISSN: 0264-6021
DT Journal
LA English
CC 2-9 (Mammalian Hormones)
AB Prostaglandins of the A type (PGA) induce the synthesis of 70 kDa **heat shock proteins** (hsp70) in a large variety of mammalian cells. Induction of hsp70 has been assocd. with a **cytoprotective** effect of PGA1 after virus infection of thermal injury. In the present report the authors provide evidence that, in murine myoblasts, PGA1 is not able to induce hsp70 expression, whereas it increases the synthesis of the constitutive protein, hsc70, and dramatically induces the synthesis of a 32 kDa protein (p32). The p32 protein has been identified as heme oxygenase. PGA1 acts at the transcriptional level by inducing heme oxygenase mRNA synthesis, and the signal for induction appears to be assocd. with decreased intracellular GSH levels. Heme oxygenase, a low-mol. mass stress protein induced in mammalian cells by oxidant stress, is known to be part of a general inducible antioxidant defense pathway. The fact that prostaglandin synthesis is stimulated in muscle during contraction and in the heart in response to ischemia raises the possibility that induction of heme oxygenase by PGA in myoblasts could be part of a protective mechanism in operation during stress and hypoxia.
ST PGA1 heme oxygenase myoblast heatshock protein
IT Myoblast
(PGA1 stimulation of heme oxygenase in myoblast independent of hsp70)
IT Proteins, specific or class
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(hsp 70, PGA1 stimulation of heme oxygenase in myoblast independent of hsp70)
IT 70-18-8, GSH, biological studies
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(PGA1 stimulation of heme oxygenase in myoblast in relation to GSH attenuation)
IT **14152-28-4**, PGA1
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(PGA1 stimulation of heme oxygenase in myoblast independent of hsp70)
IT 9059-22-7, Heme oxygenase
RL: MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(PGA1 stimulation of heme oxygenase in myoblast independent of hsp70)

=>

AN 1997:22043 CAPLUS
DN 126:112777
TI 2-Cyclopenten-1-one, a new inducer of heat shock protein 70 with antiviral activity
AU Rossi, Antonio; Elia, Giuliano; Santoro, M. Gabriella
CS Inst. Experimental Med. CNR, Viale K. Marx, Rome, 00137, Italy
SO J. Biol. Chem. (1996), 271(50), 32192-32196
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
CC 1-5 (Pharmacology)
AB The **cytoprotective** role of **heat shock proteins** (HSP) described in a variety of human diseases, including ischemia, inflammation, and infection, suggests new therapeutic strategies relying upon the development of drugs that selectively turn on heat shock genes. Cyclopentenone prostaglandins, which contain an .alpha.,.beta.-unsatd. carbonyl group in the cyclopentane ring and possess antiviral activity against several RNA and DNA viruses, were shown to function as signal for HSP synthesis in a nonstressful situation in a variety of mammalian cells. We now report that 2-cyclopenten-1-one —— selectively induces the expression of the 70-kDa HSP (HSP70) in human cells, through cycloheximide-sensitive activation of heat shock transcription factor 1 (HSF1). The .alpha.,.beta.-unsatd. carbonyl group is the key structure triggering HSF1 activation. Induction is assocd. with antiviral activity during infection with vesicular stomatitis virus. These results identify the mol. structure of natural prostaglandins responsible for HSF1 activation and open new perspectives in the search for novel antiviral and **cytoprotective** drugs.
ST antiviral cyclopentenone heat shock protein 70
IT Heat-shock factors
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(HSF1; cyclopentenone - inducer of heat shock protein 70 with antiviral activity)
IT Antiviral agents
Structure-activity relationship
(cyclopentenone - inducer of heat shock protein 70 with antiviral activity)
IT Protein HSP70
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(cyclopentenone - inducer of heat shock protein 70 with antiviral activity)
IT 111-14-8, Oenanthic acid 120-92-3, Cyclopentanone 142-29-0, Cyclopentene 3391-86-4, 1-Octen-3-ol **14152-28-4**, PGA1
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(cyclopentenone - inducer of heat shock protein 70 with antiviral activity)
IT 930-30-3, 2-Cyclopenten-1-one
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(cyclopentenone - inducer of heat shock protein 70 with antiviral activity)